WEST Search History

DATE: Monday, September 16, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L7	"p300-responsive promoter"	0	L7
L6	p300-responsive promoter	54138	L6
L5	L4 and "reporter construct"	12	L5
L4	L3 and (screen or assay)	39	L4
L3	L2 and reporter	39	L3
L2	L1 and apoptosis	64	L2
L1	p300 or cbp	856	L1

END OF SEARCH HISTORY

L2: Entry 8 of 64 File: USPT Apr 16, 2002

DOCUMENT-IDENTIFIER: US 6372739 B1
TITLE: Compounds and methods for modulation of estrogen receptors

Brief Summary Text (9):
Existing treatments for slowing bone loss generally involves administration of compounds such as estrogen, bisphosphonates, calcitonin, and raloxifene. These compounds, however, are generally used for long-term treatments, and have undesirable side effects. Further, such treatments are typically directed to the activity of mature osteoclasts, rather than reducing their formation. For example, estrogen induces the apoptosis of osteoclasts, while calcitonin causes the osteoclasts to shrink and detach from the surface of the bone (Hughes et al., Nat. Med. 2:1132-1136, 1996; (Jilka et al., Exp. Hematol. 23:500-506, 1995). Similarly, bisphosphonates decrease osteoclast activity, change their morphology, and increase the apoptosis of osteoclasts (Parfitt et al., J. Bone Miner 11:150-159, 1996; Suzuki et al., Endocrinology 137:4685-4690, 1996).

Brief Summary Text (14):
Progress over the last few years has shown that ER associates with co-activators (e.g., SRC-1, CBP and SRA) and co-repressors (e.g., SMRT and N-CoR), which also modulate the transcriptional activity of ER in a tissue-specific and ligand-specific manner. In addition, evidence now suggests that the majority of estrogen-regulated genes do not have a classical estrogen response element. In such cases, ER interacts with the transcription factors critical for regulation of these genes. Transcription factors known to be modulated in their activity by ER include, for example, AP-1, NF-.kappa.B, C/EBP and Sp-1.

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L2: Entry 7 of 64 File: USPT Apr 23, 2002

DOCUMENT-IDENTIFIER: US 6376215 B1
TITLE: JMY, a co-activator for p300/CBP, nucleic acid encoding JMY and uses thereof

Brief Summary Text (4):
By regulating the transcriptional activity of a wide variety of transcription factors, the p300/CBP family of co-activators allow diverse signals to be integrated and co-ordinated with gene expression. Significantly, p300/CBP has been implicated as a critical regulator of distinct cellular pathways, such as those leading to differentiation, cell cycle arrest and apoptosis. The molecular mechanisms that enable these processes remain unclear, although the associated histone acetyltransferase and kinase activities are likely to play an important role.

Drawing Description Text (2):
FIG. 1 shows the results of a mammalian two-hybrid assay confirming the interaction of p300 with JMY.

Drawing Description Text (3):
FIG. 2 shows that JMY interacts with two domains in p300.

Drawing Description Text (5):
FIG. 4A co-expression of JMY and p300 provides transcriptional activation of the glucocorticoid receptor (GR).

Drawing Description Text (6):
FIG. 4B shows that co-expression of JMY and p300 induce transcriptional activity of E2F-1.

Detailed Description Text (13):
Inactive portions may include fragments of the above-mentioned active portions which are capable of competing with the full length human or murine JMY protein for binding to p300. Preferably such fragments are those which are capable of antagonizing the formation of a JMY-p300 complex or JMY homodimerization, under conditions suitable for such complex formation or homodimerization to take place in the absence of such an inactive portion. Inactive portions also include dominant negative mutants of JMY.

Detailed Description Text (118):
"p300/CBP" refers to a family member of the p300/CBP family of co-activators which have histone acetyltransferase activity. p300 is described for example by Eckner et al, 1994 and CBP by Bannister and Kouzarides, 1996. For the purposes of the present invention, reference to p300/CBP refers to human allelic and synthetic variants of p300, and to other mammalian variants and allelic and synthetic variants thereof, as well as fragments of said human and mammalian forms of p300. Synthetic variants include those which have at least 80%, preferably at least 90%, homology to p300. More preferably such variants correspond to the sequence of p300 but have one or more, e.g. from 1 to 10, such as from 1 to 5, substitutions, deletions or insertions of amino acids.

Detailed Description Text (119):
Fragments of p300 and its variants are preferably at least 20, more preferably at least 50 and most preferably at least 200 amino acids in size. The p300/CBP molecule will however retain the ability to physically associate in vivo with JMY.

Detailed Description Text (141):
Alternatively, assays of the invention may be conducted by utilizing the ability of a JMY-p300/CBP complex to mediate the activation of a reporter gene or to induce a cellular response in a cell, particularly apoptosis. For example, a number of transcription factors, including the glucocorticoid receptor (GR) and E2F-1, are known to be regulated by p300/CBP, as is p53. We have found that the regulation of such factors is enhanced by JMY. We have also found that JMY is a coactivator of ER. Further, we have found that p53-mediated apoptosis is enhanced by the presence of JMY and p300. The JMY in such a complex may be in the form of a homodimer.

Detailed Description Text (146):
In an alternative embodiment, the assay may be conducted in a cell lacking wild-type p53 and which undergoes apoptosis in the presence of p53. Such cells include SAOS-2 cells. In this format the assay will be conducted by supplying to the cell expression vector(s) encoding JMY, p300/CBP and wild type p53, treating said cells with a putative modulator and measuring the effect of the modulator on apoptosis of the cells. Apoptosis may also be measured in an analogous manner in cell lines with wild type p53 wherein apoptosis is enhanced by the presence of, for example, excess JMY.

Detailed Description Text (150):
The interaction between JMY and p300 which we have established may be influenced in the cell by the actions of, inter alia, E2F-1 and pRb. It may thus be desirable to include an E2F-1, and E2F-x or other E2F family member and/or a pRb molecule in the assay of the invention. Such molecules may be included in both in vitro and in vivo assays. They may also be obtained by recombinant production, and expressed where appropriate using constructs and means analogous to those described above for the JMY and p300 molecules.

Detailed Description Text (183):
(a) The nucleic acid sequence (SEQ ID NO: 1) and primary amino acid sequence of JMY was determined (983 amino acid residues (SEQ ID NO:2). The central p300 binding domain in JMY is from residue 469 to 558. This contains hydrophobic residues that conform to a heptad leucine-rich repeat. The adenovirus E1a CR2-like motif, EVQFEILKCE (SEQ ID NO:3), is at residues 530 to 540 (residues in bold being conserved in E1a CR2). There is a proline-rich C-terminal region at residues 794-818.

Detailed Description Text (186):
JMY interacts with p300.

Detailed Description Text (187):
(a) Yeast two-hybrid assays were performed using the baits pLex-p300.sup.611-2283 or pLex-JMY.sup.469-558 with pVP16-JMY.sup.469-558 and pGAD-E1a. The results are shown in Table 1. Induction of .beta.-galactosidase (+in Table 1) indicated the major p300 interacting domain was in the central 469 to 558 segment of JMY.

Detailed Description Text (188):
(b) Co-immunoprecipitation of JMY and p300 from U2OS cells transfected with pG4 (30 .mu.g) was performed as described. After extraction, immunoprecipitation was performed with anti-Ga14 followed by immunoblotting with anti-HA (12CA5). As a control, the cell extract was immunoblotted with an anti-peptide JMY antibody in the absence or presence of competing homologous peptide. The results, which are discussed in detail below, confirmed that JMY interacts with p300 in vivo.

Detailed Description Text (191):
JMY interacts with two domains in p300

Detailed Description Text (192):
(a) Two-hybrid assay in mammalian cells were performed pG4-p300 expression vectors (1 .mu.g) were introduced into U2OS cells either alone or together with pVP16-JMY.sup.469-558 (5 .mu.g) and the reporter pG5-luc. The relative activity of luciferase to .beta.-galactosidase (derived from the internal control pCMV-.beta.gal) was determined as the average of two treatments (FIG. 2). The activity of both pG4-p300.sup.611-1257 and pG4-p300.sup.1572-2283 underwent a 4- and 5-fold increase in the presence of pVP16-JMY.sup.469-558.

Detailed Description Text (193):
(b) Binding of p300 to JMY was determined. Three different GST-p300 fusion proteins were incubated with either in vitro translated 13S E1a or JMY.sup.335-588. The three proteins were A: GST-p300.sup.1-596 ; B: GST-p300.sup.744-1571 ; and C: GST-p300.sup.1572-237. The amount of each protein bound was assessed as described below. The interaction between the 13S E1a polypeptide and GST-p300.sup.1572-2370 served as a positive control, and both GST-p300.sup.744-1571 and GST-p300.sup.1572-2370 were found to bind JMY. About 10% of the input JMY bound to each p300 fusion protein.

Detailed Description Text (195):
JMY co-activates p53 and augments apoptosis.

Detailed Description Text (196):
(a) The p53 reporter pBax-luc (2 .mu.g) together with h expression vectors for wild-type p53 (0.25 .mu.g), JMY (4 or 8 .mu.g) either alone or together with p300 (0.2 .mu.g) were transfected into SAOS2 (p53.sup.-/-) cells. Average of duplicate readings of the relative level of luciferase to the .beta.-galactosidase activity from the internal control were taken (FIG. 3).

Detailed Description Text (197):
(b) Expression vectors for p53 either alone or together with JMY were introduced into SAOS2 cells as described in the methods. Cells were fixed and treated with the anti-p53 monoclonal antibody 421 or assayed for the level of apoptosis by TUNEL. The results showed co-expression of JMY with p53 enhanced apoptosis.

Detailed Description Text (198):
(c) A quantitative comparison of the effect on apoptosis in SAOS2 cells caused by JMY, p300 or both in the presence or absence of p53 was made. The percentage of p53-positive (determined by monoclonal antibody 421) cells that were TUNEL-positive was derived, and compared to values obtained in the presence of JMY, p300, or both. The percentage increase in apoptosing cells was determined. The level of apoptosing cells caused by p53 was 17.7%, and when co-expressed with JMY apoptosis increased to 23.1%, with p300 to 16.94% and when p53 was co-expressed with JMY and p300 the level of apoptosis increased to 30.17%. The TUNEL-positive population was compared to the number of DAPI-positive cells in the absence of p53. This was used to assess the level of apoptosis in the presence of JMY and p300 which was 2.25% and 2.88% respectively. The values given were obtained from two separate assays.

Detailed Description Text (200):
JMY possesses the properties of a co-activator and co-operates with p300 in the transcriptional activation of the glucocorticoid receptor and E2F-1.

Detailed Description Text (201):
(a) The GR reporter pGRE-cat (1 .mu.g) together with expression vectors for JMY (1, 3 or 5 .mu.g) either alone or together with p300 (3 .mu.g) were transfected into HeLa cells in the absence or presence of the ligand dexamethasone (0.5 .mu.M) for the glucocorticoid receptor. The average of triplicate readings were taken to determine the relative level of CAT activity to total protein. There was significantly greater activity when both JMY and p300 were expressed together (FIG. 4a)

Detailed Description Text (202):
(b) The E2F reporter pDHFR-luc (1 .mu.g) together with expression vectors for E2F-1 (0.2 .mu.g) either alone or together with p300 (3 .mu.g) and JMY (4 and 8 .mu.g) were transfected into SAOS2 cells. The average of duplicate readings relating to the level of luciferase to .beta.-galactosidase were taken. The results showed co-expression of either JMY and p300 induce the transcriptional activity of E2F-1, and the effect is greater when both are co-expressed (FIG. 4b).

Detailed Description Text (219):
To elucidate the mechanisms of transcriptional activation by p300/CBP, we considered the possibility that additional control may be exerted through proteins that physically interact with and regulate the activity of p300/CBP. This possibility was investigated by screening for p300-interacting proteins by the yeast two-hybrid approach using a truncated p300, namely p300.sup.611-2283, fused to the LexA DNA binding domain. Although pLexA-p300.sup.611-2283 failed to activate transcription, it retained the ability to interact with a known p300/CBP-binding protein, the adenovirus E1a protein (ref.4). Using pLexA-p300.sup.611-2283 as the bait we screened a 10.5 d.p.c mouse embryo activation domain-tagged library and identified a new protein, which we have called JMY (for junction-mediating and regulatory protein).

Detailed Description Text (221):
To identify the region in JMY that is necessary for the interaction with p300/CBP, we assayed the activity of JMY derivatives as LexA hybrids in the yeast two-hybrid assay. The major p300 interacting domain in JMY was located in a 90 residue central segment, between amino acid residue 469 and 558. This same region, which contains hydrophobic residues in a heptad repeat, when assayed in the yeast two-hybrid assay can also act as a functional dimerization interface.

Detailed Description Text (222):
To determine whether p300 and JMY can interact in mammalian cells we used both immunochemical and two-hybrid based approaches. Co-transfection of U2OS cells with expression vectors encoding p300 and JMY, followed by immunoprecipitation and immunoblotting, confirmed that the interaction occurs in mammalian cells since the 110kD JMY polypeptide was present in the p300 immunoprecipitation but not the control treatment. These findings were corroborated in the mammalian two-hybrid assay in which p300 as a fusion protein with the Gal4 DNA binding domain, G4-p300.sup.611-2283, efficiently interacted with a hybrid protein containing the central domain of JMY (residues 469 to 558) fused to the VP16 transcriptional activation domain in VP16-JMY.sup.469-558 Thus, JMY and p300/CBP proteins can interact in mammalian cells.

Detailed Description Text (223):
The region in p300 that is responsible for the interaction with JMY was determine during a mammalian two-hybrid assay and a panel of p300 deletion mutants fused to the Gal4 DNA binding domain. The interaction between JMY and p300 occurred with two regions in p300, one in the C-terminal region, encompassed within residues 1572 to 2283, and the other within 611 to 1257, because when JMY-VP16 was co-expressed with either G4-p300.sup.1572-2283 or G4-p300.sup.611-1257 the transcriptional activity of the reporter pG5-luc was far more efficient than that observed in the presence of the bait alone. Other regions of p300,

such as from residue 1302 to 1572, failed to interact with JMY-VP16.

Detailed Description Text (224):
Furthermore, an interaction between JMY and p300 was evident by taking a biochemical approach in which different regions of p300 expressed as GST-fusion proteins were incubated with in vitro translated JMY. As expected, an interaction between adenovirus E1a and the C-terminal region of p300 was evident. In support of the two-hybrid assay, two regions within GST-p300.sup.744-1571 and GST -p300.sup.1572-237, bound to JMY, these same two regions overlapping with the Ga14-p300 hybrid proteins assayed earlier. These biochemical data support the conclusion that JMY interacts with two regions in p300. Thus, JMY interacts with two regions in p300, one of which resides in the C-terminal region of p300 and is known to be recognised by other regulatory proteins, including adenovirus E1a, the tumour suppressor protein p53.sup.7,8,9 and P/CAF.sup.1.

Detailed Description Text (225):
Next, we examined the possibility that JMY possessed the properties of a transcriptional regulator, firstly, by studying the effect of JMY on p53, a transcription factor which is known to be a target for p300/CBP proteins .sup.7,8,9. The transcriptional activity of p53 was assayed on the promoter taken from the Bax gene, which responds to p53 and encodes a protein that facilitates apoptosis.sup.19. The Bax promoter was efficiently induced in the presence of exogenous p53 in SAOS2 cells, cells in which the endogenous p53 gene is inactivated. A titratable increase in p53-dependent transcription was also apparent in the presence of JMY, but a further enhancement occurred when p300 was co-expressed. Similar results were observed in cell-types other than SAOS2, with the effect dependent upon the integrity of the N-terminal activations domain in p53. The transcription of other p53 target genes, such as Waf1/Cip1,.sup.22 also was enhanced by co-expression of JMY although to a lesser extent than the Bax promoter. These data indicate that JMY possesses the properties of a co-activator and, furthermore, acts together with p300/CBP proteins in the transcriptional activation of p53.

Detailed Description Text (226):
To explore the biological consequence of co-activation by JMY, we evaluated the effect of JMY on wild-type p53 activity which, when induced by genotoxic stress can, in some circumstances, cause apoptosis.sup.18,19,20,21. For This analysis we used SAOS2 cells, which are sensitive to p53-dependent apoptosis upon the introduction of wild-type p53.sup.23. Although apoptosis was evident with p53 alone, the co-expression of JMY with p53 significantly enhanced the level of apoptosis, resulting in an increase of about 30% in the total number of cells undergoing apoptosis. This influence of JMY on apoptosis was not apparent in the absence of p53, indicating that the process is dependent upon the presence of wild-type p53. In contrast however, p300 failed to affect the apoptotic activity of p53, despite causing an increase in p53-dependent transcription, although the level of p53-dependent apoptosis was significantly greater when JMY and p300 were co-expressed. These date indicate that, the induction of p53-dependent apoptosis caused by JMY can be further enhanced by p300 and, further, that an increased level of p300 alone is not sufficient to promote apoptosis.

Detailed Description Text (227):
We assessed the generality of the co-activator properties of JMY by studying the effects on two other p300/CBP-regulated transcription factors, namely the glucocorticoid receptor (GR).sup.24 and E2F-1.sup.9. In HeLa cells, where the endogenous GR functions in a ligand-dependent fashion, the co-expression of JMY or p300 potentiated the transcriptional activity of a GR-responsive promoter. As observed with p53, there was significantly greater activity when both JMY and p300 were expressed together. Thereafter, the effect of JMY on E2F-1 was explored by studying the E2F-responsive promoter taken from the DHFR gene.sup.25. The co-expression of either JMY or p300 induced the transcriptional activity of E2F-1 and, again, the effect was much greater when both JMY and p300 were introduced together compared to either alone. We conclude that JMY possessed the properties of a co-activator and, furthermore, enhances the transcriptional activity of transcription factors that respond to p300/CBP proteins.

Detailed Description Text (228):
The p300/CBP proteins are pleiotrophic co-activators that regulate a large body of transcription factors.sup.1. The mechanisms of transcriptional activation are not clear, but are likely to involve the associated histone acetyltransferase and kinase activities.sup.13,14,15,16,17, together with interactions with additional accessory molecules.sup.1,26. In This report, we have characterised a new type of co-activator that acts in concert with p300 in the activation of transcription. JMY bears no obvious similarity to other known co-activators, including those involved with nuclear receptor activation.sup.26,27,28,29, and thus likely represents a novel a class of co-activators. The protein sequence of JMY possesses a number of interesting characteristics, including homology to the adenovirus E1aCR2 motif and, although the significance of This has yet to be determined, it is noteworthy that the. interaction between JMY and p300 is sensitive to the action of adenovirus E1a.

Detailed Description Text (229):
Importantly, co-activation of p53 by JMY enhanced a specific physiological outcome, namely apoptosis, suggesting that JMY imparts an additional level of control in regulating the activity of p300/CBP responsive transcription factors. In contrast, p300 failed to effect the level of p53-dependent apoptosis, implying that JMY is an important effector molecule in directing the

cellular response to p300. For p53, the additional control provided by JMY is a significant biological determinant in regulating the physiological outcome of transcriptional activation.

Detailed Description Text (231):
Isolation of p300 interacting proteins.

Detailed Description Text (232):
The yeast strain CTY10.5 containing the LexA-.beta. galactosidase reporter vector pLex (his) was as previously described.sup.30. pLex-p300.sup.611-2283 was made by subcloning the Ndel fragment of p300 (3028 to 8046bp) into the Sall site of pLex (his). Screening a 10.5 d.p.c. mouse embryo random primed cDNA library fused to the VP16 trans activation domain.sup.31 yielded a single positive clone containing 280bp of JMY sequence. Full length JMY cDNAs were isolated through a combined approach of screening cDNA libraries prepared from F9 EC.sup.32 and PCC4 mouse teratocarcinoma (Stratagene) cells and RACE (Clontech).

Detailed Description Text (236):
The activity of p(GRE).sub.2 TATA-Cat (1.0 .mu.g) was assayed in HeLa cells after transfection of the indicated amounts of p300 and JMY expression vectors (pCMV-p300 and pCMV-JMY). After transfection, cells were incubated in medium containing 10% dextran coated charcoal stripped serum with or without 0.5 .mu.M dexamethasone for 24h, when cells were harvested and assayed for CAT activity. For transfection into SAOS2 cells, cells were incubated in 10% serum throughout and transfected with either pBax-luc.sup.19 (2.0 .mu.g) or pDHFR-luc.sup.25 (1.0 .mu.g) with either the expression vector for p53 (pCMV-p53; 0.25 .mu.g) or E2F-1 (pCMV-E2F-1; 0.2 .mu.g) respectively together with the indicated amounts of pCMV-p300 and PCMV-JMY and harvested 24-36h post transfection. All transfections were performed using the calcium phosphate procedure and included an-internal control pCMV-.beta.-gal.

Detailed Description Text (239):
Three regions of p300 were expressed as GST fusion proteins, namely GST-p300.sup.1-596, -p300.sup.744-1571 and -p300.sup.1572-2370, as described.sup.2. Purified fusion proteins were incubated with in vitro translated JMY.sup.335-588 or the E1a 13S polypeptide for 1h at 4.degree. C. and then washed in 50mTris pH8.0, 150 mM NaCl and 0.1% NP40. The remaining bound protein proteins were separated on a 10% SDS-PAGE gel and detected by autoradioaugraphy. For immunoprecipitation, U2OS were transfected with expression vectors pG4-p300.sup.611-2283, pG4, or pCMV-JMY (containing an HA epitope) by the calcium phosphate procedure. After 48h, cells were harvested in 50 mM Tris-HCl pH7.4, 60 mM NaCl, 5 mM EDTA, 0.5% NP40, 50 mM NaF, 1 mM DTT, 1 mM PMSF, 0.2 mM sodium orthovanedate, leupeptin (0.5 .mu.g/ml), protease inhibitor (0.5 .mu.g/ml), trypsin inhibitor (1.0 .mu.g/ml) aprotinin (0.5 .mu.g/ml) and bestatin (40 .mu.g/ml) and incubated on ice for 30 min. The cell extract was pre-cleared by incubating with protein G agarose for 30 min at 4.degree. C., and the supernatant immunoprecipitated with anti-mouse Gal4 monoclonal antibody (Santa-Cruz) which was harvested with protein-A agarose. The agarose beads were collected and washed three times in the extraction buffer before denaturation and SDS-polyacrylamide (7.5%) gel electrophoresis. Immunoblotting was subsequently performed with an anti-mouse HA monoclonal antibody (Boehringer Mannheim) or an anti-peptide rabbit antibody against a peptide taken from JMY.

Detailed Description Text (240):
Immunofluorescence and apoptosis assays.

Detailed Description Text (241):
SAOS2 cells, grown in 10% foetal calf serum, were transfected with pCMV-p53 (3 .mu.g) either alone or together with pCMV-JMY (5 .mu.g), pCMV-p300 (5 .mu.g) or both. After 14h incubation, cells were washed and further incubated for 24h in the presence of 0.2% serum. Cells were fixed in 4% paraformaldehyde at 4.degree. C. for 20 min, rinsed and permeabilised in PBS containing 0.2% Triton X-100 at 4.degree. C. for 10 min. Subsequently, calls were treated with the anti-p53 monoclonal antibody 421, washed and further incubated in tetramethylrhodamine-conjugated goat anti-mouse (Southern Biotechnology Associates, Inc.) for 2h at room temperature. For the TUNEL (TdT-mediated dUTP nick end labelling) analysis, cells were incubated in a Ca.sup.2+ reaction buffer containing fluorescein-dUTP and dNTP, and terminal deoxynucleotidyl transferase (Boehringer Mannheim) at 37.degree. C. for 1h. For DAPI (4', 6-diamidine-2-phenylindole) staining, cells were incubated with DAPI (0.2 .mu.g/ml) in PBS at room temperature for 2 min. Coverslips were washed three times in PBS, mounted and viewed.

Detailed Description Text (251):
9. Lee, C-W., Sorensen, T. S., Shikama, N. and La Thangue, N. B. Functional interplay between p53 and E2F through co-activator p300; Submitted.

Other Reference Publication (12):
Avantaggiati et al., "SV40 large T antigen and adenovirus E1a oncoproteins interact with distinct isoforms of the transcriptional co-activator, p300," The EMBO Journal, 15(9):2236-2248, 1996.

Other Reference Publication (13):
Bannister et al., "The CBP co-activator is a histone acetyltransferase," Nature, 384:841-643, 1996.

Other Reference Publication (30):
Gu et al., Synergistic activation of transcription by CBP and p53, Nature, 387:819-822, 1997.

Other Reference Publication (32):
Hanstein et al., "p300 is a component of an estrogen receptor coactivator complex," Proc. Natl. Acad. Sci. USA, 93:11540-11545, 1996.

Other Reference Publication (42):
Lundblad et al., "Adenoviral E1A-associated protein p300 as a functional homologue of the transcriptional co-activator CBP," Nature, 374:85-88, 1995.

Other Reference Publication (46):
Muraoka et al., "p300 gene alterations in colorectal and gastric carcinomas," Oncogene, 12:1565-1569, 1996.

Other Reference Publication (49):
Ogryzko et al., "The Transcriptional Coactivators p300 and CBP Are Histone Acetyltransferases," Cell, 87:953-959, 1996.

Other Reference Publication (51):
Perkins et al., "Regulation of NF-.kappa.B by Cyclin-Dependent Kinases Associated with the p300 Coactivator," Science, 275:523-527, 1997.

Other Reference Publication (52):
Puri et al., "p300 is required for MyoD-dependent cell cycle . . . ," J. EMBO, 16(2):369-383, 1997.

Other Reference Publication (53):
Sabbatini et al., "Essential role for p53-mediated transcription in E1 A-induced apoptosis," Genes & Develop., 9:2184-2192, 1995.

Other Reference Publication (57):
Torchia et al., "The transcriptional co-activator p/CIP binds CBP and mediates nuclear-receptor function," Nature, 387:677-684, 1997.